CS7-02 Management of Hepatitis C Virus Infection in Patients with Chronic Renal Failure and/or Thalassaemia

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Concurrent Session 8 – Gastrointestinal Infections

CS8-01 Norovirus Infection: An Emerging Enteric Infection

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CS8-02 *Blastocystis*-Host Interactions: New Insights on Pathogenesis

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Aims: *Blastocystis* is an anaerobic, enteric, protistan parasite with zoonotic potential. Its clinical relevance has been debated extensively, as some studies support a pathogenic role for the parasite while others show no correlation between infection and disease. Humans can be infected by a variety of distinct genotypes (subtypes). We hypothesize that subtypes vary in pathogenic potential and we aimed to investigate if such differences can be observed using *in vitro* cytopathic assays.
Methods & Results: Investigations were carried on *Blastocystis* subtypes 4 and 7, which are zoonotic genotypes found in rodent and avian hosts respectively. Cysteine protease activity was determined by azocasein assay. The results revealed inter- and intra-subtype variations in protease activity, with subtype 7 isolates harboring higher cysteine protease levels. Apoptosis assays for phosphatidylserine (PS) externalization and nuclear blebbing revealed that subtype 7 induced more cell death in host cells than subtype 4. Similarly, transwell assays for barrier function showed that subtype 7 induced greater barrier disruption than subtype 4. A stress fiber model developed in Caco-2 colonic epithelial cells, revealed that subtype 7 induced reorganization of stress fibers with a possible link to barrier function compromise.
Conclusions: From this study, clear cytopathic differences exist between *Blastocystis* subtypes, which suggest that variations in pathogenic potential exists among *Blastocystis* genotypes from different animal hosts.
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CS8-03 A Novel and Dominant Serotype *Shigella flexneri* Fxb Emerging in China

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CS8-04 Clinical Manifestations and Management of Enterovirus 71 Brain Stem Encephalitis

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Several epidemic outbreaks of Enterovirus 71 (EV71) infection occurred during the past one decade in southern Taiwan. EV71 has the potential to cause a large outbreak worldwide such as that in Taiwan in 1998. The main clinical presentations are herpangina, hand-foot-and-mouth disease (HFMD), and central nervous system (CNS) complications. Brain stem encephalitis (BE) is the cardinal feature of EV71 CNS complications during the outbreak. The predominant neurological presentations are myoclonus jerk, vomiting, and ataxia. BE that progressed abruptly to neurogenic shock and pulmonary edema (PE) was indicative of poor prognosis and high mortality. EV71 BE was categorized into uncomplicated BE, autonomic nervous system (ANS) dysregulation, and PE by disease severity. The PE that occurs in children with EV71 BE is caused by abnormal cytokines activation that produces severe CNS and systemic inflammatory responses. Currently, there is no specific antiviral agent to treat or vaccine to prevent EV71 diseases. Intravenous immunoglobulin (IVIG) has been found to have broad therapeutic applications for the treatment of many infectious diseases. We found a decrease in the plasma concentration of various cytokines following administration of IVIG. Patients with ANS dsyregulation is the critical timing to received IVIG infusion. It is possible that a more favorable survival might have been obtained by earlier therapy and larger doses of IVIG. Milrinone, cyclic nucleotide phosphodiesterase inhibitor subtype III, increases cardiac output, and reduces systemic vascular resistance and pulmonary capillary wedge pressure without excessive increases in myocardial oxygen consumption. EV71-associated PE patients treat with milrinone is associated with significantly decreased mortality by attenuated sympathetic activity and cytokine production. Controlled clinical trials are ongoing to confirm these observations. A better understanding of the clinical features and management of EV71 BE may shed light on improving the outcome of severe and complicated EV71 BE.

Concurrent Session 9 – HBV: Treatment Options and Strategies

CS9-01 The Role of Currently Available Antiviral Drugs in Hepatitis B

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The main role of antiviral drug therapy for chronic hepatitis B (CHB) infection is to suppress the hepatitis B virus (HBV) from active replication, thereby preventing hepatitis and the complications. Doctors and patients alike wished for achieving HBV eradication early. This will lower the risk of liver complications and restore health and quality of life of the CHB patients. From the healthcare provider point of view, the transmission of HBV infection can be minimized and the health cost substantially reduced. Eradication of HBV rarely occurs spontaneously; around 0.5 to 2% among CHB individuals lost HBsAg annually. Newer antiviral drugs have higher efficacy and safety. HBsAg loss over and above the estimated spontaneous incidence has been reported after and during therapy. The role of current available antiviral drugs in the full spectrum of CHB disease can be summarized as follows.
There are two main categories of antiviral therapy for HBV infection: (1) interferon-based injections, and (2) oral nucleos(t)ide analogues. Any HBV disease types with viraemia will benefit from antiviral therapy that is efficacious, safe and affordable. Most international and national HBV treatment guidelines focused on individuals with HBV DNA level over 5 $\log_{10}$ copies/ml (i.e. around 4 $\log_{10}$ IU/L); especially if HBeAg negative; associated with raised serum ALT; and have evidence of significant liver fibrosis. Antiviral therapy is particularly important among males, over middle age, has family history of cirrhosis and hepatocellular carcinoma (HCC). The aim is to halt the existing liver damage, hopefully reverse fibrosis, even cirrhosis, and eliminate the conditions that favour HCC development. Clinical experience and data do support such strategy.